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Case Report

Dengue fever with encephalitis: An uncommon presentation of common illness

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ABSTRACT

The dengue virus is a type of Flavivirus that causes dengue fever. It is mostly found in tropical and subtropical countries, with Southeast Asia bearing the brunt of the disease burden. The virus can affect a wide range of organ systems, and the disease can range from a mild flu-like illness to severe dengue hemorrhagic fever or dengue shock syndrome. We present a case of a 21-year-old female who presented with dengue fever with neurological manifestations. Findings from a brain MRI, electroencephalogram, CT scan, and some routine investigations were supported our diagnosis of dengue encephalitis. Conservative and supportive care was provided, including intravenous fluid therapy, antibiotics, antivirals, antiseizure medications, and corticosteroids. The patient's GCS gradually improved with treatment, and physical therapy was also provided. Treatment that is both effective and timely can reduce associated morbidity and result in complete recovery.

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1. Introduction

Dengue fever and dengue hemorrhagic fever are becoming more common among India's young people.¹ This is caused by Flaviviridae genes flavivirus, a family of positive SS RNA viruses.² The virus that causes dengue has four closely related but antigenically distinct serotypes: DEN-1, 2, 3, and 4. Dengue has traditionally been thought to be non-neurotropic, but the most commonly involved serotypes in neurotropism are DEN-2 and DEN-3.³ Dengue encephalopathy is a secondary manifestation of dengue fever that is frequently associated with multisystem derangement discovered later in the course of the illness and is thought to be an immune-mediated inflammatory response of the body.⁴ On the other hand, encephalitis is caused by a direct virus invasion.⁵ We present a case of dengue encephalitis with unusual MRI findings.

The uncommon presentation of this common illness creates diagnostic quandaries and can delay standard treatment, resulting in unfavorable outcomes. Reporting these presentations and understanding the disease's epidemiological patterns aids physicians in arriving at the correct diagnosis at the right time.

2. Case Presentation

A 21-year-old female presented with fever, vomiting and body ache for past 4 days; with a single attack of severe headache and generalized tonic clonic seizure followed by unconsciousness for 6 hours. On clinical examination temperature was 98.5-degree F, Pulse rate was 56 b/m, Blood Pressure was 100/60 mmHg, Oxygen saturation was 99% at room air and random blood sugar was 28 mg%. There was no history of any active bleed. On physical examination pallor, icterus, cyanosis and edema were absent. GCS was E₂V₁M₃, pupils were bilaterally equal and

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reacting to light, Babinski reflex was present. Cardiovascular and Respiratory system was within normal limits.

2.1. Investigations

The initial Complete Blood Count depicts hemoglobin of 10.6 g/dl, platelet counts 26,000/mcl, and WBC 10,320/mm³. Serum electrolytes report shows Na⁺ 142.5meq/dl, K⁺ 3.8 meq/dl, Cl⁻ 109.3 mmol/l and serum Ca²⁺ was 7.6 mg/dl. The Serum creatinine level was 0.6 mg/dl, SGOT was 310 IU/L, and SGPT was 130 IU/L. Dengue NS1 antigen, dengue antibody IgG and IgM were found reactive, scrub typhus antibody was positive. Typhi dot IgG, IgM and rapid malaria antigen tests were found negative. CT scan head showed "bilateral thalamus appears hypodense". CSF analysis revealed normal pressure, clear and colourless fluid, TLC 04/cmm (all lymphocytes), protein 69 mg/dl, sugar 44mg/dl and was negative for tuberculosis. Electroencephalogram finding was suggestive of global cerebral dysfunction.

The MRI brain with Gd contrast shown hyper intensities on T2w and flare images with patchy restricted diffusion in both sides of mid brain, pons and thalamo capsuloganglionic region with subacute stage hemorrhagic foci in bilateral thalami S/O hemorrhagic encephalitis.

2.2. Treatment

A presumptive diagnosis of viral encephalitis with scrub typhus was made and she was started on Intravenous antibiotics meropenem sulbactam 1.5 gm 8 hourly, injection doxycycline 100 mg 12 hourly, injection levetiracetem 500mg 8 hourly, injection dextrose 25% 8 hourly, injection calcium gluconate 6 hourly, injection pantoprazole 40 mg xOD, injection ondansetron 4 mg 12 hourly, and intravenous fluid NS 500 ml 8 hourly.

Supportive care with nasogastric feeding, and urinary catheterization were provided and she was continuously monitored in the intensive care unit to identify clinical or biochemical deterioration. After 2 days of this initial treatment injection acyclovir 500 mg 8 hourly started with tablet citicoline & piracetam twice daily on 3rd day of admission through nasogastric tube.

Acyclovir was given for 14 days; antibiotics and PPI till corticosteroid treatment, tab. levetiracetem, tab citicoline & piracetam were tapered and stopped over the period of 2 months.

2.3. Progress and follow up

After one day of treatment platelets fallen to 23000/mcl and all other finding were within normal limits. Clinically patient GCS was same as previous E₂V₂M₃. After 2 days of admission SGOT/SGPT started falling and platelets started to increase (71,000/mcl), hematocrit was 42.4%. Clinically patient started opening eye spontaneously but

no improvement in verbal and motor commands. On 4th days of treatment platelets were 68,000/mcl and hematocrit was 31.7%. SGOT/SGPT further fallen to 91/103 U/L respectively.

On 5th days of treatment GCS was improved E₄V₂M₃ and Laboratory findings started to become in normal range (platelets 1,43,000/mcl). On 6th day of treatment patient started moving her limbs and GCS score started to improve (E₄V₂M₄) slowly.

After 9th days of admission patient was shifted from ICU to general ward with GCS score of E₄V₂M₅ and she was started on injection dexamethasone 4 mg thrice daily for 3 days, twice a day for next 3 days, and once daily for next 3 days with antibiotics and proton pump inhibitors for support. Then she was brought on oral corticosteroids (tab. Prednisolone) 20 mg, 10 mg, 5 mg each for 7 days and then stopped. Patient showed marked improvement in GCS within 7 days of corticosteroid treatment.

Patient started walking independently and GCS became E₄V₅M₆ but ataxia was present, patient could not walk in straight line and Romberg sign was positive which slowly improved with further treatment. Patient was discharged after 3 weeks from hospital with GCS E₄V₅M₆ and oral medications.

After 2 months of follow up, she showed remarkable recovery of her neurologic function without any residual weakness but minimal ataxia was present with supervised physiotherapy.

3. Discussion

Dengue fever is the most prevalent viral infection transmitted by arthropods in humans.² Dengue fever offers a wide range of infection consequences (asymptomatic to symptomatic). Undifferentiated fever (viral syndrome), DF, DHF, and dengue with atypical symptoms such as isolated organopathies are examples of symptomatic disease.³ Encephalitis and meningoencephalitis have been observed in 4-21% of dengue cases.⁶ Many cases of dengue encephalitis and other neurological signs have been described by Misra et al (India),⁷ Kularatne et al (Sri Lanka),⁸ and Solomon et al (Vietnam).⁹ They all exhibited neurological symptoms such as headaches, disorientation, seizures, hemiparesis, and even coma. Some of them described EEG and MRI alterations based on the location of the brain affected. Carod-Artal et al¹⁰ presented the findings of eight autopsy studies on fatal dengue cases with neurological involvement that revealed histopathologic patterns of cerebral edoema, congestion, bleeding, perivascular lymphocytes infiltration, and even necrosis of brain matter. There are several case reports and research in the literature that demonstrate how various areas of the brain can be impacted in various ways. Our patient's brain MRI revealed hyperintensities on both sides of the midbrain, pons, and thalamo capsuloganglionic area, as well

as subacute stage hemorrhagic foci in bilateral thalami.

The majority of cases reported in the literature were based on a clinical diagnosis of encephalitis, followed by serological confirmation of dengue infection and exclusion of other neurotropic viruses. Although MRI or CT scans are nonspecific, they can provide valuable signals for additional investigations including CSF testing. As with this case, it can be difficult to suspect dengue encephalitis based on history, examination, and preliminary testing. This type of unusual DF presentation might result in diagnostic delays and poor patient outcomes. This case report underscores the need of evaluating dengue as a differential diagnosis in a patient with encephalitis symptoms, especially in the context of a dengue epidemic and endemicity, as it modifies surveillance and care. In an uncommon presentation like this, a combination of clinical symptoms and other basic examinations such as PLT, WBC, PCV, and liver enzymes would provide clues to diagnosis and careful patient monitoring to prevent the potentially fatal complication of dengue sickness. Another key point to remember is that, while there are national recommendations for managing DF, each patient's management plan must be individualized to their specific needs.

4. Conclusion

This patient was diagnosed with severe dengue with central nervous system symptoms that were not accompanied by shock, usual signs, or bleeding. As a result, in endemic dengue locations, health practitioners should consider infections by this virus in patients with fever and neurological manifestations as part of the initial diagnosis, and they should be on the lookout for neurological abnormalities that may occur after acute dengue sickness.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare no conflict of interest.

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